Published Studies on Health Effects of Woodsmoke

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ABSTRACT

This paper summarises published health effects of woodsmoke a) in developing countries from heating or cooking with wood, b) in developed countries, mainly from domestic heating using modern wood stoves, c) area-wide health effects, such as declines in lung function or increased hospital admissions, observed when woodsmoke builds up because of topographical conditions in places where heating with modern woodstoves is common and d) results of exposing laboratory animals to woodsmoke, such as changes in immune function, mortality after exposure to woodsmoke plus a bacteriological challenge, or mortality in rats with bronchitis exposed to concentrations of PM2.5 particles not dissimilar to levels measured in areas with a woodsmoke problem. The consistent pattern, from nearly all the 45 papers listed, is that exposure to woodsmoke can result in significant, deleterious health effects.

DEVELOPING COUNTRIES

Exposure to woodsmoke can have serious consequences for health. In developing countries, some stoves may emit smoke to indoor air. Several studies have shown that exposure to woodsmoke reduces lung function, especially in children, and increases coughs and other respiratory diseases. In Bogota, woodsmoke exposure may explain about half of all cases of obstructive airways disease. In Mexico City, famous for its traffic pollution, women exposed to woodsmoke had 3.9 times the risk of chronic bronchitis and 9.7 times the risk of chronic bronchitis plus chronic airway obstruction. If exposed for an average of 33 mins or more a day (200 or more hours/year), risks were 15 and 75 times higher than in women not exposed to woodsmoke. In Africa, cooking with wood increased the risk of stillbirth by 50%. Homes with woodstoves were found to have greater concentration of mutagenic material in the air resulting in increased risk of cancer. These results are summarised in Table 1.

DEVELOPED COUNTRIES

Many recent studies have also found significant effects in woodstove users in developed countries such as the US. Children from homes with woodstoves tend to have increased respiratory symptoms, acute lower respiratory infection, increased frequency and severity of wheeze, increased frequency

Table 1 - Symptoms from woodstove use in developing countries.
Direct quotes from publications are italicised

Reference	Symptoms in households using wood heat or cooking in developing countries
Dennis et al., 1996.	Conclusions: *'This study showed that among elderly women of low socioeconomic status in Bogota, woodsmoke exposure is associated with the development of Obstructive Airways Disease (OAD) and may help explain around 50% of all OAD cases.'*
Ellgard, 1996.	*'The association between exposure to air pollution from cooking fuels and health aspects was studied in Maputo, Mozambique. Almost 1,200 randomly selected women residing in the suburbs of Maputo were interviewed … Wood users were found to have significantly more cough symptoms than other groups. This association remained significant when controlling for a large number of environmental variables.'*
Gharaibeh, 1996.	A significant negative impact was found with regard to environmental exposure to both passive smoking and wood and kerosene unvented cooking stoves. Reduction in lung function for wood and kerosene were FVC (L): 1.02-1.32; FEV(1) (L): 0.91-1.25; FEF(25-75) (L/S): 1.24-1.86; PEFR (L/S): 1.67-2.64. Primary school children in Jordan.
Guneser et al., 1994.	*'Pulmonary function levels were diminished in passive smokers and in children whose houses were heated by a wood-burning stove. As a result, passive smoking, using a wood-burning stove for heating, and family history of respiratory diseases are to be considered risk factors for the respiratory system.'* 617 primary school children, aged 9-12, studied in Turkey.
Hamada et al., 1992.	*'Strikingly higher ($p < 0.01$) levels of polycyclic aromatic hydrocarbons, and much higher ($p = 0.07$) levels of uspended particulate matter were found in the kitchens with wood stoves. These findings appear to support the hypothesis that domestic wood burning stoves are risk factors for some upper digestive and respiratory tract cancers in Brazil.'*
Perez-padilla et al., 1996.	*'A case-control study was performed in women older than 40 yr of age in Mexico City to evaluate the risk of cooking with traditional wood stoves for chronic bronchitis and chronic airway obstruction (CAO). Crude odds ratios for wood smoke exposure were 3.9 (95% CI, 2.0 to 7.6) for chronic bronchitis only, 9.7 (95% CI, 3.7 to 27) for CAO plus chronic bronchitis, and 1.8 (95% CI, 0.7 to 4.7) for CAO … The findings support a causal role of domestic wood smoke exposure in chronic bronchitis and chronic airflow obstruction.'*
Sandoval et al., 1993.	Describes *'the clinical, radiologic, functional, and pulmonary hemodynamic characteristics of a group of 30 nonsmoking patients with a lung disease that may be related to intense, long-standing indoor wood-smoke exposure… Fibrous and inflammatory focal thickening of the alveolar septa as well as diffuse parenchymal anthracotic deposits are the most prominent pathologic findings … Pulmonary arterial hypertension in wood-smoke inhalation-associated lung disease appears to be more severe than in other forms of interstitial lung disease and tobacco-related COPD.'* Authors' address: Mexico City.
Ardayfio Schandorf, 1993.	*'cooking over an open fuel wood stove resulted in an almost 50% greater chance of stillbirth among pregnant women.'* Rural Africa.
Zhang et al., 1996.	*'Among the 6 fuel/stove combinations, wood stove generated the highest cancer risk and LPG generated the least risk … The cancer risk of benzene or that of styrene from use of biomass cookstoves might exceed the risk from all sources of airborne benzene or styrene in the US.'* Manila, the Philippines.

Table 2 - Studies in the US and other developed countries of symptoms associated with households using wood heat

Reference	Respiratory and other symptoms in households using wood heat
Honicky et al., 1985.	Moderate and severe respiratory symptoms were significantly greater (P<.001) in 34 children, aged 1-7 years in houses with woodstoves than in 34 children houses without. Conclusion: *'Present findings suggest that indoor heating with wood-burning stoves may be a significant etiologic factor in the occurrence of symptoms of respiratory illness in young children.'* Michigan, US.
Butterfield, et al., 1989.	Significant correlation (P<.01), between woodstove use and frequency of wheeze, severity of wheeze, frequency of cough and waking up at night with cough, based on 59 subjects aged 1 to 5.5 years.
Lipsett et al., 1991.	Presence of woodstove or fireplace in the home was associated with shortness of breath in females and both shortness of breath and moderate or severe cough in males (p<0.01 for all cases). 182 asthmatics living in Denver, Colorado.
Betchley et al., 1997	Forest firefighters had significant declines in lung function (FEV(1)). Declines, pre-shift to midshift, averaged 0.089 L, 0.190 L, and 0.439 L/sec in TVC, FEV(1) and FEF (25-75). The use of wood for indoor heat also was associated with the declines in FEV(1).
Morris et al., 1990.	58 Navajo children under 2 years with diagnosed pneumonia or bronchiolitis were compared with matched control children. Use of a wood burning stove was associated with a four times higher risk of lower respiratory tract infection (P<.001).
Robin et al., 1996.	Study of 45 Navajo children aged less than 2 years and hospitalised with Acute Lower Respiratory Infection (ALRI) and 45 matched controls. *'Indoor air concentration of respirable particles was positively correlated with cooking and heating with wood (P < 0.02) but not with other sources of combustion emissions. Conclusions. Cooking with wood-burning stoves was associated with higher indoor air concentrations of respirable particles and with an increased risk of ALRI in Navajo children.'*
Tuthill, 1984.	Risk of respiratory symptoms increased by 10%, but this was not statistically significant. Study of children aged 5-11, 258 with woodstoves, 141 without. Exposure to formaldehyde from any source, including wood burning, significantly increased risk.
Daigler et al., 1991.	A comparison of patients in New York with physician-diagnosed otitis media (n = 125, 74% response), and Controls (n = 237, 72% response) showed exposure to a woodburning stove was significantly associated (P<.05 with increased otitis (an inflammation of the middle ear marked by pain, fever, dizziness, and abnormalities of hearing).
Hogg, 1997.	The author comments on the case report by Dr. David T. Janigan and colleagues of classic bronchiolitis obliterans in a man who used a wood-burning stove to dispose of construction materials in Canada.
Dean et al., 1992.	Case of methemoglobinemia, sudden onset of cyanosis, irritability, metabolic acidosis, and a lethal methemoglobin level of 71.4% in a 10 week old infant. Family history revealed a wood-burning stove which emitted pine tar fumes as the potential environmental methemoglobin-producing source. The infant's cradle was situated five feet from the stove. The baby was treated and recovered.
Ramage et al., 1988.	Case study of 61-yr-old woman suffering shortness of breath on exertion and interstitial lung disease. Bronchoalveolar lavage revealed numerous carbonaceous particulates and fibers, as well as cellular and immunoglobulin abnormalities. Inflammation and fibrosis were found surrounding them on open biopsy. The particle source was traced to a malfunctioning wood-burning heater in the patient's home.
Von Mutius et al., 1996.	Children in Bavarian homes heated with wood had less hayfever and were less sensitive to pollen, cold air or other irritants. Postulated that wood heating may be more common in farming or country areas where children are more likely to be exposed, and hence desensitised, to pollens eg from haymaking.
Volkmer et al., 1995.	*'The use of a wood fire/heater compared to other forms of heating was significantly associated with a reduced prevalence rate for dry cough (OR 0.84) and ever having wheezed (OR 0.82).'* Findings contradict most other studies. Data not adjusted for confounders such as socio-economic status, except by area code.
van Houdt et al., 1986.	*'The use of wood stoves caused an increase of indoor mutagenicity in 8 out of 12 homes.'*
Boone et al., 1989.	*'Woodsmoke proved to be a major source of indirect genotoxins in homes. The increase is probably due to higher concentrations of polycyclic aromatic hydrocarbons in the wood smoke aerosol …'* USA.
Alfheim et al., 1984.	*'Whereas wood heating in an "airtight" stove was found to cause only minor changes in the concentration of PAH and no measurable increase of mutagenic activity of the indoor air, both these parameters increased considerably when wood was burned in an open fireplace, yielding PAH concentrations comparable to those of ambient urban air. Woodburning in the closed stove did, however, result in increased concentrations of mutagenic compounds and PAH on particles sampled in the vicinity of the house.'*

of cough and waking up at night with a cough. Measured lung function has also been found to decline (See Table 2). In addition, forest firefighters in the US had decreased lung function after fighting fires. Use of wood for indoor heat by these firefighters was also associated with decreased lung function. Van Houdt *et al*. (1986) found increased indoor mutagenicity in 8 out of 12 homes using woodstoves. In New York, woodstove use was found to be significantly associated (P<.05) with increased otitis media (an inflammation of the middle ear marked by pain, fever, dizziness, and abnormalities of hearing). Studies detecting adverse health effects in families using wood heating are listed in Table 2 as well as three studies showing increased mutagenicity or genotoxity of the air inside, or in the vicinity of, homes using wood heating. Three case studies, from North Carolina, Pennsylvania and Canada, describe illnesses attributed specifically to fumes from woodstoves.

Very few studies have found little or no effect of exposure to woodsmoke or woodstove use. Exceptions are one study in Bavaria (Von Mutius *et al.*, 1996) which found that wood or coal heating was associated with a reduced incidence hay fever and decreased sensitisation to pollen or other aeroallergens. The authors postulated this may have been due to such heating being associated with farming or country areas, where children were more likely to be exposed to pollens eg from haymaking. A South Australian study (Volkmer *et al*., 1995) found parental smoking was associated with decreased eczema and use of evaporative cooling was

associated with increased dry cough. Woodstove use was also associated with many factors. However, when the data for Adelaide were considered by themselves, most of these apparent effects were no longer significant and there was little difference between woodstove use and flued natural gas. Both had reduced incidence of dry cough ($P < .05$) compared with electric heating. Flued gas users had significantly reduced incidence of wheeze in the previous 12 months; woodstove users had reduced incidence of ever wheezing. Conversely, unflued gas heating increased the incidence of dry cough and wheeze.

The Volkmer study was not, however, adjusted for socio-economic factors except on an area-wide basis. Some associations might therefore be explained by differences between city and country areas in heating, cooling, smoking or other factors. In addition, some of the methodological problems which led to the apparent associations in the State-wide analysis may well remain in the data for Adelaide, explaining other unusual associations. An intriguing alternative hypothesis is that respiratory tract infection during early childhood may confer protection against sensitisation to aeroallergens (Holt *et al*., 1997; Strachan, 1989). Washing hands 3 or more times a day was also associated with increased risk of asthma in a study in Bristol, UK, reported in newspapers and TV news bulletins. If immune systems need priming in early childhood to reduce the risk of allergic reactions, it may be possible to devise prevention strategies based on controlled exposure at the appropriate age to weakened or benign agents. Increasing the risk of infection by reduced cleanliness or exposure to woodsmoke might have a similar effect, but at the cost of increased risk for everyone else, including existing asthmatics, the elderly and babies, for whom particulate pollution has been linked to cot deaths (Woodruff *et al*., 1997).

As woodstoves become more air-tight, it is also possible that neighbours downwind of the stove receive at least as much noxious pollution as the family using the stove, making such effects easier to detect on an area-wide basis, rather than by type of heating used by individual families. Alfheim *et al*. (1984) found the effect on the mutagenicity of the air of burning wood in an airtight stove was in the vicinity of the house, rather than in the room with the stove. Table 3 shows how use of woodstoves increases health risk for entire cities or areas.

Table 3 - Studies relating outdoor concentrations of woodsmoke to adverse health effects in the whole population

Reference	Adverse Effects of Outdoor Woodsmoke
Schwartz, 1993.	Significant association between visits to 8 hospital emergency departments in Seattle for asthma and PM10 pollution. In 1993, wood burning was found to be the dominant source of PM10 pollution in Seattle in all seasons of the year, ranging from 60% in summer to 90% in winter.
Koenig et al., 1993.	Significant association in Seattle (where the majority of particulate air pollution originates from woodsmoke) between outdoor fine particle pollution and decreased lung function (measured by spirometry) in asthmatic children aged 8-11.
Heumann et al., 1991.	Children with the highest exposure to wood smoke had a significant decrease in lung function, measured by FEV1 and FVC. 410 children aged 8-11 in Klamath Falls, Oregon.
Johnson, 1990.	Particle pollution from woodsmoke in the air was associated with significant decreases in lung function in children aged 8-11. 495 subjects in Montana.
Browning, et al.,1990.	No statistically significant differences, but a pattern of increased symptoms and chronic illness in children aged 1- 5 in the area with high wood smoke.
Lipsett et al., 1997.	'*These results demonstrate an association between ambient wintertime PM10 and exacerbations of asthma in an area where one of the principal sources of PM10 is Residential Wood Combustion.'* Santa Clara County, California.
Betchley, et al.,1997.	Forest firefighters had significant declines in lung function (FEV(1)). Average declines, pre-shift to midshift were 0.089 L, 0.190 L, and 0.439 L/sec in TVC, FEV(1) and FEF (25- 75). The use of wood for indoor heat also was associated with the declines in FEV(1).
Morgan et al., 1998.	In Sydney, Australia, death rates are generally higher in winter and increase on days of, or days following high particle pollution. Particle concentrations are generally higher in winter. Carbon dating of air EPA NSW, 1996 samples at Rozelle, near the CBD, in July and August 1993, found that for % from wood two-thirds of particles originated from wood, not coal, oil or diesel. At Winmalee in the Blue Mountains, the proportion was 80%. Samples were taken from 4 pm to 8 am the following morning at times when the air was essentially free of smoke from bushfires or hazard reduction burns. Solid fuel use in Sydney increased from 7% of households in 1988 to 13% in 1995. The majority of winter particle pollution in Sydney is thus caused by a small minority of households using wood heaters.
Lewtas et al., 1991.	Mutagenicity testing of air containing smoke emitted from woodheaters in Boise, Idaho, US, using the Ames test on salmonella and tumor initiation assays in mice, found that woodsmoke was 12 times more carcinogenic than an equal concentration of cigarette smoke.
Larson & Koenig, 1994.	*'We conclude that the preponderance of the data suggest a causal relationship between elevated wood smoke levels and adverse respiratory health outcomes in young children.'*

CITY-WIDE DETRIMENTAL HEALTH EFFECTS FROM WOODSTOVE USE IN SOME HOUSEHOLDS

In areas with airtight stoves, the smoke is vented to the outside air. Neighbours living downwind probably suffer greater exposure. Whole neighbourhoods have been found to suffer as a result. For example, in Seattle, where 90% of winter particulate air pollution was found to originate from woodsmoke, hospital admissions for asthma were found to increase on days with high pollution. Similar effects were observed in Santa Clara County, where residential wood burning is also a principal source of particulate air pollution. Separate studies in Klamath Falls, Oregon and in Montana found declines in children's lung function when ambient levels of woodsmoke were high. Declines in lung function with woodsmoke exposure were also found for asthmatic children in Seattle. In Sydney, the majority of particle pollution in winter arises from less than 13% of households using wood heaters (EPA NSW, 1996). Death rates and particle pollution are generally higher in winter and particle pollution on a daily basis was found to be associated with increased death rates (Morgan *et al*., 1998). In Boise, Idaho, US, air containing smoke emitted from woodheaters was tested for mutagenicity using the Ames test on salmonella and tumour initiation assays in mice. Woodsmoke was estimated to be 12 times more carcinogenic than an equal concentration of cigarette smoke (Lewtas *et al*.,1991).

LABORATORY EVIDENCE

Mounting laboratory evidence is now available to explain many of these adverse effects. Lal *et al*. (1993) describe in detail some of the nasty effects seen in lungs of rats exposed to woodsmoke. Other researchers have found deleterious effects

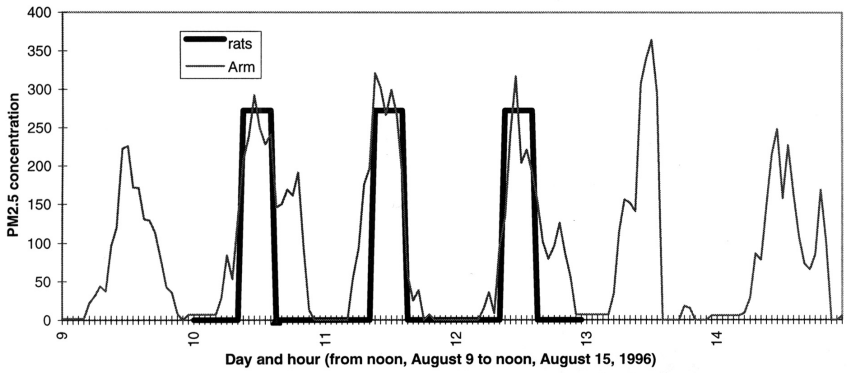
Table 4 - Physiological measurements of effects of exposure to woodsmoke

Reference	Physiological measurements of effects of exposure to woodsmoke
Stone, 1995.	Mice were exposed for 6 hours to wood smoke, emissions from an oil furnace or no pollution (control) and then an aerosol of the bacterium *Streptococcus zooepidemicus,* which causes severe respiratory infections. After 2 weeks, 5% of the mice in the control group exposed to air and bacteria had died, along with a similar percentage of the mice breathing the oil fumes. But 21% of the wood-smoked mice were felled.
Stone, 1995.	Rats were exposed to no pollution, or 800 µg/m^3 wood smoke for 1 hour, then to golden staph bacteria. The bacteria were more virulent in animals which breathed the woodsmoke. This was attributed to a suppression in activity of the rats' macrophages, immune cells that roam the body, looking to engulf and destroy foreign particles.
Kou et al. , 1997.	*'These results suggest that an increase in OH burden following smoke inhalation is actively involved in evoking the acute irritant effects of wood smoke on breathing in rats.'*
Rao et al., 1995.	Metabolites of woodsmoke condensate accumulate in cultured rat eye lenses, compromising ability to accumulate rubidium-86 (mimic of K) and choline. Says may explain implication of smoke in cataract.
Lal et al., 1993.	Rats exposed to woodsmoke suffered *'bronchiolitis, hyperplasia and hypertrophy of bronchiolar epithelial lining cells, some necrosed lining cells desquamated into lumens, congestion of parenchymatous blood vessels, oedema, hyperplasia of lymphoid follicles, peribronchiolar and perivascular infiltration of polymorphonuclear cells, and mild emphysema'* Conditioned worsened with accumulated exposure.*'The results indicate progressive pathomorphological pulmonary lesions with subsequent exposure to wood smoke in controlled conditions.'*
Churg et al., 1997.	Autopsies were carried out of lung tissue from 10 never-smoking long-term residents of Vancouver. Retained particles in human lung parenchyma were counted, sized, and identified by analytical electron microscopy. 96% of particles had aerodynamic diameter less than 2.5 microns.
Godleski et al., 1996.	Rats with bronchitis were exposed for 6 hours per day to 272 ug/m^3 PM2.5. 37% of rats exposed to particles died, compared to none exposed to filtered air.

after only 1 hour's exposure to 800 µg/m^3 of woodsmoke. Following exposure, bacteria in rats' lungs were more virulent (Stone, 1995). In Australia, woodsmoke concentrations can approach this level and remain high for several hours. For example, in Armidale, NSW, our measurements using a TEOM in one residential area showed hourly woodsmoke have peaked at 467 µg/m^3 and remained over 200 µg/m^3 for more than 10 hours. Another experiment (Stone, 1995) subjected mice either to woodsmoke, oil furnace fumes, or clean air for 6 hours. Mice were then challenged by an aerosol of the bacterium *Streptococcus zooepidemicus*. 21% of the mice exposed to wood smoke were dead two weeks later, compared with only 5% mice exposed to fumes from the oil furnace or to clean air. The article comments: *'Part of the problem is that wood smoke is a witch's brew of carcinogens, including aldehydes and polycyclic aromatic hydrocarbons, carbon monoxide, and organic particles less than 10 microns in diameter, called PM10. PM10s have been implicated in increased morbidity and mortality on days of heavy air pollution.'*

Very recent research has found that only the smaller particles less than 2.5 microns are linked with mortality and morbidity. Schwartz et al. (1996) analysed daily mortality in six Eastern US Cities over 8 years. Particles between 2.5 and 10 microns had no association with daily mortality. The strongest association was with PM2.5s (particles less than 2.5 microns in diameter) which arise mainly from combustion. Woodsmoke is almost entirely PM2.5s. (Larson and Koenig, 1994). Autopsies have shown that particles less than 2.5 microns in diameter are retained in human lungs, but not larger particles (Churg *et al*., 1997). Godleski *et al*. (1996) exposed rats with bronchitis for 6 hours per day to 272 µg/m^3 PM2.5. 37% of rats exposed to particles died, compared to none exposed to filtered air. In terms of air quality standards based on daily averages, 272 µg/m^3 PM2.5 over 6 hours would be equated to a daily average of 68 µg/m^3. Similar or worse PM2.5 concentrations are often observed in areas where woodsmoke builds up during night time inversions. For example, Figure 1 compares PM2.5 concentrations recorded hourly by a TEOM in a residential area of Armidale, NSW from 9 to 14 August 1996, with the rats' exposure. Overall, our measurements show daily average PM2.5 concentrations in this residential area of Armidale exceeded 68 µg/m^3 on 29% and 38% of nights in July-August 1996 and June-August 1997.

Figure 1 - Comparison of PM2.5 exposure (µg/m^3) in Godleski's rats experiment with woodsmoke pollution measured using a TEOM in a residential area of Armidale from August 9 to 15, 1996.



IMPLICATIONS

More than 40 studies relating woodsmoke to health are referenced here. They cover all aspects, from use of wood for cooking or heating in developing countries, use of modern heaters in developed countries, to detrimental health effects found in whole towns or cities when woodsmoke is allowed to build up. The epidemiological evidence is supported by tests on laboratory animals, showing exposure to woodsmoke reduces the ability of the lungs to fight infection. After exposure to a pathogen, 21% of mice exposed to woodsmoke became sick and died within 2 weeks, compared with 5% exposed to oil furnace fumes. The size and the consistency of detrimental health effects and the relatively large proportion of total winter pollution (the majority of particles in Sydney come from heating less than 13% of households; EPA NSW, 1996) indicates that serious consideration should be given to the cost effectiveness of much more stringent controls on woodheaters and encouragement of alternatives.

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Abstracts of most of these papers are on the world wide web. See http://lash.une.edu.au/~drobinso/smog_refdescr.htm

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